Refine Search

Search Results -

Terms	Documents
(u6 and h1) same promoter$	219

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L3

Refine Search

Recall Text | Clear | Interrupt

Search History

DATE: Wednesday, May 17, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L3	(u6 and h1) same promoter$	219	L3
DB=USPT; PLUR=NO; OP=OR			
L2	L1 and promoter$	1	L2
L1	6506559.pn. or 5801154.pn.	2	L1

END OF SEARCH HISTORY

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n the RD set.
      S3       67  RD  (unique items)
? s s3 and (treat? or patient)
Processing
Processed  10 of  40 files ...
Processing
Processed  30 of  40 files ...
Completed processing all files
              67  S3
        17202100  TREAT?
         5942154  PATIENT
      S4       24  S3 AND (TREAT? OR PATIENT)
? rd

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.
      S5       24  RD  (unique items)
? show files;ds;t/3,k/all
File   5:Biosis Previews(R) 1969-2006/May W1
         (c) 2006 BIOSIS
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File   8:Ei Compendex(R) 1970-2006/May W1
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         (c) 2005 The HW Wilson Co.
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         (c) 2006 The HW Wilson Co.
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File 136:BioEngineering Abstracts 1966-2006/Apr
         (c) 2006 CSA.
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         (c) 2006 The HW Wilson Co
File 144:Pascal 1973-2006/Apr W3
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         (c) format only 2006 Dialog
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         (c) 2006 Elsevier Science B.V.
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         (c) 2006 DECHEMA
File 357:Derwent Biotech Res. _1982-2006/May W1
         (c) 2006 Thomson Derwent & ISI
File 358:Current BioTech Abs 1983-2006/Jan
          (c) 2006 DECHEMA
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File 369:New Scientist 1994-2006/Mar W1
(c) 2006 Reed Business Information Ltd.
File 370:Science 1996-1999/Jul W3
(c) 1999 AAAS
File 399:CA SEARCH(R) 1967-2006/UD=14421
(c) 2006 American Chemical Society
File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
(c) 1998 Inst for Sci Info
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File 41:Pollution Abstracts 1966-2006/Apr
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Set	Items	Description
S1	0	(SIRNA OR RNAI) (S) (HEPATITIS C)
S2	85	(SIRNA OR RNAI) AND (HEPATITIS C)
S3	67	RD (unique items)
S4	24	S3 AND (TREAT? OR PATIENT)
S5	24	RD (unique items)

>>>KWIC option is not available in file(s): 399

5/3,K/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0015765184 BIOSIS NO.: 200600110579
Inhibition of hepatitis C virus replication by pol III-directed overexpression of RNA decoys corresponding to stem-loop structures in the NS5B coding region
AUTHOR: Zhang Jing (Reprint); Yamada Osamu; Sakamoto Takashi; Yoshida Hiroshi; Araki Hiromasa; Murata Takayuki; Shimotohno Kunitada
AUTHOR ADDRESS: FUSO Pharmaceut Ind LTD, Ctr Res and Dev, Joto Ku, 2-3-30 Morinomiya, Osaka 5368523, Japan**Japan
AUTHOR E-MAIL ADDRESS: j-zhang@fuso-pharm.co.jp
JOURNAL: Virology 342 (2): p276-285 NOV 25 2005 2005
ISSN: 0042-6822

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: RNA-based approach to inhibit a wide range of HCV isolates. These results suggest that SL RNA decoys may prove to be useful in the *treatment* of hepatitis C, which may be advantageous over other sequence-specific gene therapy modalities (such as antisense RNA and *siRNA*) in preventing the escape of genetic variants. (c) 2005 Elsevier Inc. All rights reserved.
DESCRIPTORS:
DISEASES: *hepatitis C*...

5/3,K/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0015495952 BIOSIS NO.: 200510190452
Activation of the N-Ras-PI3K-Akt-mTOR pathway by hepatitis C virus: Control of cell survival and viral replication
AUTHOR: Mannova Petra; Beretta Laura (Reprint)
AUTHOR ADDRESS: Fred Hutchinson Canc Res Ctr, Div Publ Hlth Sci, 1100 Fairview Ave N,M5-A864,Bpx 19024, Seattle, WA 98109 USA**USA
AUTHOR E-MAIL ADDRESS: lberetta@fhcrc.org
JOURNAL: Journal of Virology 79 (14): p8742-8749 JUL 2005 2005
ISSN: 0022-538X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: apoptosis. We also characterized the role of this pathway in HCV replication. Reduction of N-Ras expression by transfection of N-Ras small interfering RNA (*siRNA*) resulted in increased replication of HCV. We observed a similar increase in HCV replication in cells *treated* with the PI3K inhibitor LY294002 and in cells transfected with mTOR *siRNA*. Taken together, these data suggest that increased N-Ras levels in subcellular sites of HCV replication and stimulation of the prosurvival PI3K-Akt pathway and...
DESCRIPTORS:
DISEASES: *hepatitis C*...
CHEMICALS & BIOCHEMICALS: ...small interfering RNA {*siRNA*};

5/3,K/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0015224813 BIOSIS NO.: 200500131450
Characterization of host-range and cell entry properties of the major genotypes and subtypes of hepatitis C virus
AUTHOR: Lavillette Dimitri; Tarr Alexander W; Voisset Cecile; Donot Peggy; Bartosch Birke; Bain Christine; Patel Arvind H; Dubuisson Jean; Ball Jonathan K; Cosset Francois-Loic (Reprint)
AUTHOR ADDRESS: LVRTGINSERMU412,IFR128,BioSci Lyon Gerland, Ecole Normale Super Lyon, 46 Allee Italie, F-69364, Lyon, 07, France**France
AUTHOR E-MAIL ADDRESS: flcosset@ens-lyon.fr
JOURNAL: Hepatology 41 (2): p265-274 February 2005 2005
MEDIUM: print
ISSN: 0270-9139 _(ISSN print)
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: and neutralization properties of parental HCV. Because sequence variations in E1 and E2 may account for differences in tropism, replication properties, neutralization, and response to *treatment* in patients infected with different genotypes, we investigated the functional properties of HCV envelope glycoproteins from different genotypes/subtypes. Our studies indicate that hepatocytes were...

...in CD81-deficient HepG2 cells indicated that CD81 is used by all the different genotypes/subtypes analyzed to enter the cells. However, by silencing RNA (*siRNA*) interference assays, our results show that the level of Scavenger Receptor Class-B Type-I (SR-BI) needed for efficient infection varies between genotypes and...
DESCRIPTORS:
DISEASES: *hepatitis C*...
...METHODS & EQUIPMENT: silencing RNA interference assay {*siRNA* interference assay...

5/3,K/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014901824 BIOSIS NO.: 200400272581
Hepatitis C virus induces a mutator phenotype: Enhanced mutations of immunoglobulin and protooncogenes
AUTHOR: Machida Keigo; Cheng Kevin T-N; Sung Vicky M-H; Shimodaira Shigetaka; Lindsay Karen L; Levine Alexandra M; Lai Ming-Yang; Lai Michael M C (Reprint)
AUTHOR ADDRESS: Keck Sch MedDept Mol Microbiol & Immunol, Univ So Calif, 2011 Zonal Ave, Los Angeles, CA, 90033, USA**USA
AUTHOR E-MAIL ADDRESS: michlai@hsc.usc.edu
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 101 (12): p4262-4267 March 23, 2004 2004
MEDIUM: print
ISSN: 0027-8424 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

DESCRIPTORS:
...ORGANISMS: host, *patient*
...DISEASES: *hepatitis C*
CHEMICALS & BIOCHEMICALS: ...small interfering RNA {*siRNA]*}

5/3,K/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014764732 BIOSIS NO.: 200400132086
Development of *siRNA* as a therapeutic for hepatitis C.
AUTHOR: Morrissey David V (Reprint); Jensen Kristi (Reprint); Zinnen Shawn (Reprint); Dickinson Brent (Reprint); Hartsough Kim (Reprint); Shaw Cindy (Reprint); McSwiggen James A (Reprint); Vargeese Chandra (Reprint); Bowman Keith (Reprint); Shaffer Chris S (Reprint); Polisky Barry (Reprint); Lockridge Jennifer A (Reprint)
AUTHOR ADDRESS: Sirna Therapeutics, Inc., Boulder, CO, USA**USA
JOURNAL: Hepatology 38 (4 Suppl. 1): p627A October 2003 2003

MEDIUM: print
CONFERENCE/MEETING: 54th Annual Meeting of the American Association for the Study of Liver Diseases Boston, MA, USA October 24-28, 2003; 20031024
SPONSOR: American Association for the Study of Liver Diseases
ISSN: 0270-9139 _(ISSN print)
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

Development of *siRNA* as a therapeutic for hepatitis C.

ABSTRACT: RNA interference (*RNAi*) is a recently discovered cellular mechanism that recognizes and destroys double-stranded RNA, and appears to play a role in the cell's anti-viral defense system. Short interfering RNA molecules (*siRNA*) are approximately 21 nucleotide long, double-stranded RNA intermediates of the *RNAi* mechanism that guide unique *RNAi* protein machinery to the target RNA. This machinery, known as RISC (RNA induced silencing complex) then degrades the target RNA. To develop synthetic *siRNA* molecules as therapeutic agents for systemic administration in vivo, a panel of chemical modifications designed to enhance physical stability was introduced into chemically synthesized siRNAs...

...significantly prolonged stability in serum and human liver extracts. Cell culture studies in the HCV subgenomic replicon system revealed a high degree of inhibition following *treatment* with these chemically modified siRNAs. Most importantly, the degree of inhibition seen with the chemically modified, stabilized siRNAs was similar to that seen with all-RNA (unmodified) *siRNA* molecules. In addition, prolonged duration of *siRNA*-induced gene silencing was observed with chemically stabilized siRNAs in comparison to all-RNA molecules. Efficient in vivo delivery of the stabilized synthetic *siRNA* to the liver is also being addressed by direct conjugation of targeting ligands to HCV-specific siRNAs. Preliminary pharmacokinetic studies have been completed for three...
...after a subcutaneous injection with 5% of the dose found intact in the liver. These stabilized and targeted compounds may provide new options in the *treatment* of hepatitis.
DESCRIPTORS:
DISEASES: *hepatitis C*...
CHEMICALS & BIOCHEMICALS: short interfering RNA {*siRNA]*}

5/3,K/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014679118 BIOSIS NO.: 200400059875
Identification of the most accessible sites to ribozymes on the hepatitis C virus internal ribosome entry site.
AUTHOR: Ryu Kyung-Ju; Lee Seong-Wook (Reprint)
AUTHOR ADDRESS: Department of Molecular Biology, Dankook University, Seoul, 140-714, South Korea**South Korea
AUTHOR E-MAIL ADDRESS: SWL0208@unitel.co.kr
JOURNAL: Journal of Biochemistry and Molecular Biology 36 (6): p538-544 November 30, 2003 2003
MEDIUM: print
ISSN: 1225-8687
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: hepatitis C virus (HCV) is a major causative agent of chronic hepatitis and hepatocellular carcinoma. The development of alternative antiviral therapies is warranted because current *treatments* for the HCV infection affect only a limited number of patients and lead to significant toxicities. The HCV genome is exclusively present in the RNA form; therefore, ribozyme strategies to target certain HCV sequences have been proposed as anti-HCV *treatments*. In this study, we determined which regions of the internal ribosome entry site (IRES) of HCV are accessible to ribozymes by employing an RNA mapping...

...regions of target RNAs and have important implications for the development of various antiviral therapies which are based on RNA such as ribozyme, antisense, or *siRNA.*
DESCRIPTORS:
DISEASES: *hepatitis C*...
CHEMICALS & BIOCHEMICALS: ...*siRNA* {small interfering RNA}

5/3,K/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014473207 BIOSIS NO.: 200300428051
Inhibition of intracellular hepatitis C virus replication by synthetic and vector-derived small interfering RNAs.
AUTHOR: Yokota Takanori (Reprint); Sakamoto Naoya; Enomoto Nobuyuki; Tanabe Yoko; Miyagishi Makoto; Maekawa Shinya; Yi Li; Kurosaki Masayuki; Taira Kazunari; Watanabe Mamoru; Mizusawa Hidehiro
AUTHOR ADDRESS: Department of Neurology and Neurological Science, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo, 113-8519, Japan**Japan
AUTHOR E-MAIL ADDRESS: tak-yokota.nuro@tmd.ac.jp; nsakamoto.gast@tmd.ac.jp
JOURNAL: EMBO Reports 4 (6): p602-608 June 2003 2003
MEDIUM: print
ISSN: 1469-221X _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

...ABSTRACT: for siRNAs. Importantly, we have identified an effective site in the 5' UTR at which apprx80% suppression of HCV replication was achieved with concentrations of *siRNA* as low as 2.5 nM. Furthermore, DNA-based vectors expressing *siRNA* against HCV were also effective, which might allow the efficient delivery of *RNAi* into hepatocytes in vivo using viral vectors. Our results support the feasibility of using *siRNA*-based gene therapy to inhibit HCV replication, which may prove to be valuable in the *treatment* of hepatitis C.
DESCRIPTORS:
DISEASES: *hepatitis C*...
METHODS & EQUIPMENT: small interfering RNA-based gene therapy {*siRNA* -based gene therapy...

5/3,K/8 (Item 1 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002793499 IP ACCESSION NO: 6684315
Enhanced Sensitivity of Human Hepatoma Cells to 5-Fluorouracil by Small Interfering RNA Targeting Bcl-2

Kanda, Tatsuo; Yokosuka, Osamu; Imazeki, Fumio; Arai, Makoto; Saisho, Hiromitsu
Department of Medicine and Clinical Oncology, Graduate School of Medicine, Chiba University, 1-8-1 Inohana, Chuo-ku, Chiba 260-0856, Japan, [mailto:kandat-cib@umin.ac.jp]

DNA and Cell Biology, v 24, n 12, p 805-809, December 2005
PUBLICATION DATE: 2005

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 1044-5498
FILE SEGMENT: Nucleic Acids Abstracts

ABSTRACT:
... the apoptosis induced in human hepatocellular carcinoma (HCC) cell lines by 5-fluorouracil (5-FU) could be enhanced by transfecting Bcl-2 small interfering RNA (*siRNA*). Bcl-2 *siRNA* and control *siRNA* were transfected into cells following *treatment* with or without 5-FU. Suppression of Bcl-2 expression was confirmed by Western blotting; cell viability was evaluated by MTS assay, and the occurrence of apoptosis in cells was evaluated by apoptosis assay. Expression of Bcl-2 protein after transfection of 20 nM Bcl-2 *siRNA* was significantly lower than that of control. Incubation of all cell lines with Bcl-2 *siRNA* reduced cell viability 96 h after 5-FU *treatment* compared with all other controls: Huh-7 ($P < 0.01$), Huh-7 with hepatitis C replicon ($P < 0.01$), HepG2 ($P < 0.01$), HLE ($P < 0.05$). Moreover, the proportion of apoptosis in control *siRNA*, Bcl-2 *siRNA*, control *siRNA* prior to 5-FU *treatment*, and Bcl-2 *siRNA* prior to 5-FU *treatment* groups were (4.6 plus or minus 2.3)%, (7.5 plus or minus 0.5)%, (6.0 plus or minus 2.1)%, and (19.5 plus or minus 0.86)%, respectively. The Bcl-2 *siRNA* prior to 5-FU *treatment* group showed the strongest effect of inducing apoptosis. In conclusion, the combination Bcl-2 *siRNA* and 5-FU might represent a new therapeutic option for HCC.

DESCRIPTORS: *siRNA*; Bcl-2 protein; Apoptosis; 5-Fluorouracil; Tumor cell lines; Transfection; *Hepatitis C*; Hepatoma; Western blotting; Hepatocellular carcinoma

5/3,K/9 (Item 2 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts

0002723184 IP ACCESSION NO: 6276003
Hepatitis C Virus Replicons Escape RNA Interference Induced by a Short Interfering RNA Directed against the NS5b Coding Region

Wilson, Joyce A; Richardson, Christopher D
Ontario Cancer Institute/University Health Network, 620 University Ave. Suite 706, Toronto, Canada M5G 2C1. Department of Medical Biophysics, University of Toronto, 610 University Ave., Toronto, Canada M5G 2M9

Journal of Virology, v 79, n 11, p 7050-7058, June 1, 2005
PUBLICATION DATE: 2005

PUBLISHER: American Society for Microbiology, 1752 N Street N.W. Washington, DC 20036 USA, [URL:http://www.asm.org/]

DOCUMENT TYPE: Journal Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English
ISSN: 0022-538X
FILE SEGMENT: Virology & AIDS Abstracts; Nucleic Acids Abstracts
ABSTRACT:
RNA interference represents an exciting new technology that could have therapeutic applications for the *treatment* of viral infections. Hepatitis C virus (HCV) is a major cause of chronic liver disease and affects over 270 million individuals worldwide. The HCV genome...

...single-stranded RNA that functions as both an mRNA and a replication template, making it an attractive target for therapeutic approaches using short interfering RNA (*siRNA*). We have shown previously that double-stranded *siRNA* molecules designed to target the HCV genome block gene expression and RNA synthesis from hepatitis C replicons propagated in human liver cells. However, we now show that this block is not complete. After several *treatments* with a highly effective *siRNA*, we have shown growth of replicon RNAs that are resistant to subsequent *treatment* with the same *siRNA*. However, these replicon RNAs were not resistant to *siRNA* targeting another part of the genome. Sequence analysis of the *siRNA*-resistant replicons showed the generation of point mutations within the *siRNA* target sequence. In addition, the use of a combination of two siRNAs together severely limited escape mutant evolution. This suggests that RNA interference activity could be used as a *treatment* to reduce the devastating effects of HCV replication on the liver and the use of multiple siRNAs could prevent the emergence of resistant viruses.

DESCRIPTORS: *siRNA*; Genomes; RNA-mediated interference; Replication; Evolution; Liver diseases; *Hepatitis C*; Gene expression; Hepatocytes; Point mutation; Therapeutic applications; Transcription; Infection; Hepatitis C virus

5/3,K/10 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

13764761 EMBASE No: 2006186489
Destroying RNA as a therapeutic approach
Tafech A.; Bassett T.; Sparanese D.; Lee C.H.
C.H. Lee, Chemistry Program, University of Northern British Columbia, 3333 University Way, Prince George, BC V2N 4Z9 Canada
AUTHOR EMAIL: leec@unbc.ca
Current Medicinal Chemistry (CURR. MED. CHEM.) (Netherlands) 2006, 13/8 (863-881)
CODEN: CMCHE ISSN: 0929-8673
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 168

...Such approaches can be used in the study of gene function as in functional genomics, in the identification of disease-associated genes, and for the *treatment* of human diseases. This review provides a comprehensive up-to-date look at all the current available technologies used for the destruction of RNA, with...
MANUFACTURER NAMES: Isis/United States; Genta; *Sirna*/United States; Hybridon
MEDICAL DESCRIPTORS:

...effect--side effect--si; in vivo study; in vitro study; gene silencing; gene expression regulation; Huntington chorea; graft rejection --complication--co; graft rejection--prevention--pc; *hepatitis C*; cardiovascular disease; Alzheimer disease; cell based gene therapy; drug half life; nonviral gene delivery system; viral gene delivery system; adenovirus vector; drug potency; drug...

5/3,K/11 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

13764576 EMBASE No: 2006178943
Assessing evidence from clinical trials in chronic hepatitis C
Bacon B.R.
Dr. B.R. Bacon, Division of Gastroenterology and Hepatology, Saint Louis University Liver Center, Saint Louis University School of Medicine, 3635 Vista Avenue at Grand Blvd., St Louis, MO 63110 United States
AUTHOR EMAIL: baconbr@slu.edu
Journal of Viral Hepatitis (J. VIRAL HEPATITIS) (United Kingdom) 2006 , 13/SUPPL. 1 (1-5)
CODEN: JVHEE ISSN: 1352-0504 eISSN: 1365-2893
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 40

...often not well tolerated and is giving rise to a growing number of nonresponders. As a result, a large number of experimental drugs for the *treatment* of chronic hepatitis C are in development. As the clinical trial reports are made available, physicians need to become familiar with issues related to the...
...BRAND NAME/MANUFACTURER NAME: albuferon/Human Genome Sciences/United States; actimmune/Intermune/United States; viramidine/Valeant/United States ; cellcept/Hoffmann La Roche/United States; isis14803/Isis/United States; heptazyme/*Sirna*/United States; biln 2061/Boehringer Ingelheim/Germany; hcv 371/Wyeth/United States
...MANUFACTURER NAMES: States; Valeant/United States; Astellas/Japan; Human Genome Sciences/United States; Intermune/United States; Intarcia/United States; Hoffmann La Roche/United States; Isis/United States; *Sirna*/United States; Boehringer Ingelheim/Germany; Wyeth/United States
MEDICAL DESCRIPTORS:
**hepatitis C*--drug therapy--dt; **hepatitis C*--etiology--et
...virus; remission; drug tolerability; evidence based medicine; clinical assessment; methodology; flu like syndrome--side effect--si; depression --side effect--si; hemolytic anemia--side effect--si; *patient* compliance; cardiotoxicity--side effect--si; drug efficacy; human; clinical trial; review; priority journal

5/3,K/12 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

13747319 EMBASE No: 2006169689
Antisense *treatments* for biothreat agents
Warfield K.L.; Panchal R.G.; Aman M.J.; Bavari S.
S. Bavari, US Army Medical Research Institute of Infectious Diseases, Fort Detrick, Frederick, MD 21702 United States
AUTHOR EMAIL: sina.bavari@amedd.army.mil
Current Opinion in Molecular Therapeutics (CURR. OPIN. MOL. THER.) (United Kingdom) 2006, 8/2 (93-103)

CODEN: CUOTF ISSN: 1464-8431
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 128

Antisense *treatments* for biothreat agents

Antisense oligomers (ASOs) represent a promising technology to *treat* viral and bacterial infections, and have already been shown to be successful against a variety of pathogens in cell culture studies and nonhuman primate models of infection. For these reasons, antisense technologies are being pursued as *treatments* against biothreat agents such as Ebola virus, dengue virus and Bacillus anthracis. Several generations of modified oligonucleotides have been developed to maximize nuclease resistance, target...
BRAND NAME/MANUFACTURER NAME: ogx 011/Oncogenex; *sirna* 027/*Sirna*; *sirna 027*/Allergan; isis 5132; isis 2503; GEM 231
MANUFACTURER NAMES: Genta; Oncogenex; *Sirna*; Allergan
MEDICAL DESCRIPTORS:
...structure; drug efficacy; drug mixture; gene silencing; nonviral gene therapy; enzyme activation; gene expression; in vitro study; in vivo study; drug distribution; drug blood level; *hepatitis C*--drug therapy--dt; Hepatitis C virus; liver cancer--drug therapy--dt; enzyme inhibition; oncogene H ras; complement activation; side effect--side effect--si; cardiovascular...
...DRUG TERMS (UNCONTROLLED): drug administration--iv; phosphorothioate oligodeoxynucleotide derivative--subcutaneous drug administration--sc; ogx 011--pharmacology--pd; methylphosphonate oligodeoxynucleotide derivative --pharmacology--pd; 2' o methyloligoribonucleotide derivative--pharmacology --pd; *sirna* 027

5/3,K/13 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

13240582 EMBASE No: 2005305050
Small interfering RNA effectively inhibits protein expression and negative strand RNA synthesis from a full-length hepatitis C virus clone
Prabhu R.; Vittal P.; Yin Q.; Flemington E.; Carry R.; Robichaux W.H.; Dash S.
S. Dash, Department of Pathology and Laboratory Medicine, Tulane University Health Sciences Center, 1430 Tulane Avenue, New Orleans, LA 70112 United States
AUTHOR EMAIL: sdash@tulane.edu
Journal of Medical Virology (J. MED. VIROL.) (United States) 2005, 76/4 (511-519)
CODEN: JMVID ISSN: 0146-6615
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 59

Hepatitis C virus (HCV) infection is usually *treated* with the combination of interferon and ribavirin, but only a small fraction of patients develop a sustained remission. There is need for the development of specific molecular approaches for the *treatment* of chronic HCV infection. We propose that RNA interference is highly effective antiviral strategy that offers great potential for the *treatment* of HCV infection. Three plasmid constructs expressing small interfering RNAs (siRNAs) targeted to sequences encoding the structural gene (E2) and non-structural genes (NS3, NS5B...

...as shown by ribonuclease protection assay (RPA). All three siRNAs efficiently inhibited synthesis of replicative negative strand HCV RNA in the transfected cells. A control *siRNA* plasmid against a Epstein-Barr virus latency gene did not inhibit protein expression and negative strand HCV RNA. These results suggest that *RNAi* is an effective and alternative approach that can be used to inhibit HCV expression and replication. (c) 2005 Wiley-Liss, Inc.
MEDICAL DESCRIPTORS:
hepatitis C--etiology--et; molecular cloning; protein expression; protein targeting; gene replication; genetic transcription; Adenovirus; Western blotting; immunocytochemistry; immunization; ribonuclease protection assay; plasmid; Epstein Barr virus...

5/3,K/14 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

13073281 EMBASE No: 2005138461
Biochemical prevention and *treatment* of viral infections - A new paradigm in medicine for infectious diseases
Le Calvez H.; Yu M.; Fang F.
H. Le Calvez, Abgent, Inc., 6310 Nancy Ridge Drive, San Diego, CA 92121 United States
AUTHOR EMAIL: lecalvez@abgent.com
Virology Journal (VIROL. J.) (United Kingdom) 23 NOV 2004, 1/-
ISSN: 1743-422X
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 51

Biochemical prevention and *treatment* of viral infections - A new paradigm in medicine for infectious diseases

...significant portion of the population. In the recent years, FDA's approval and subsequent market acceptance of Synagis, a monoclonal antibody indicated for prevention and *treatment* of respiratory syncytial virus (RSV) has heralded a new era for viral infection prevention and *treatment* . This emerging paradigm, herein designated "Biochemical Prevention and *Treatment*", currently involves two aspects: (1) preventing viral entry via passive transfer of specific protein-based anti-viral molecules or host cell receptor blockers; (2) inhibiting viral amplification by targeting the viral mRNA with anti-sense DNA, ribozyme, or RNA interference (*RNAi*). This article summarizes the current status of this field. (c) 2004 Le Calvez et al; licensee BioMed Central Ltd.
MEDICAL DESCRIPTORS:
...pneumovirus; drug efficacy; retinitis--drug therapy--dt; cancer--drug therapy--dt; psoriasis--drug therapy--dt; Crohn disease--drug therapy--dt; ulcerative colitis--drug therapy--dt; *hepatitis C*--drug therapy--dt; solid tumor--drug therapy--dt; asthma--drug therapy--dt; rheumatoid arthritis--drug therapy--dt; restenosis--drug therapy--dt; kidney polycystic disease...

5/3,K/15 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

12533513 EMBASE No: 2004126713
Therapeutic potential of retroviral *RNAi* vectors

Devroe E.; Silver P.A.
P.A. Silver, Dept. Biol. Chem./Molec. Pharmacol., Harvard Medical School, Boston, MA 02115 United States
AUTHOR EMAIL: pamela silver@dfci.harvard.edu
Expert Opinion on Biological Therapy (EXPERT OPIN. BIOL. THER.) (United Kingdom) 2004, 4/3 (319-327)
CODEN: EOBTA ISSN: 1471-2598
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 100

Therapeutic potential of retroviral *RNAi* vectors

The ability of small interfering RNA (*siRNA*) to mediate gene-specific posttranscriptional silencing in mammalian cells will undoubtedly revolutionise functional genomics, as well as drug target identification and validation. Furthermore, there is widespread excitement that *siRNA* itself might prove useful in the clinical setting. For those wishing to develop *siRNA* as a therapeutic agent, the most difficult obstacle to overcome will be delivery. Recently, several breakthroughs have highlighted viruses as excellent vehicles for *siRNA* delivery. Retroviruses, the transgene-delivery vector of choice for many experimental gene therapy studies, have been engineered to deliver and stably express therapeutic *siRNA* within cells, both in vitro and in vivo. These findings are important milestones for the development of *siRNA* as a gene therapy for *treatment* of viral infections, cancer, autoimmune syndromes and numerous genetic disorders. This review describes the development of retroviral *siRNA* vectors, highlights proof-of-concept experiments demonstrating their therapeutic efficacy and explores therapeutic targets particularly suitable for retroviral-mediated gene silencing.
MEDICAL DESCRIPTORS:
gene silencing; Human immunodeficiency virus infection--drug therapy--dt; cancer; virus hepatitis; transgene; protein expression; gene therapy; autoimmune disease; genetic disorder; hepatitis B; *hepatitis C*; Lentivirinae; side effect--side effect--si; human; nonhuman; review

5/3,K/16 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

12291262 EMBASE No: 2003403709
Silence of the genes: A targeted approach to the suppression of specific genes in human disease using small interfering RNA (*siRNA)*
Apostolopoulos J.
J. Apostolopoulos, Centre of Inflammatory Diseases, Monash University, Department of Medicine, 246 Clayton Road, Clayton, Vic. 3168 Australia
AUTHOR EMAIL: jimja@unimelb.edu.au
Current Genomics (CURR. GENOMICS) (Netherlands) 2003, 4/7 (587-598)
CODEN: CGUEA ISSN: 1389-2029
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 101

Silence of the genes: A targeted approach to the suppression of specific genes in human disease using small interfering RNA (*siRNA)*

The ability to silence specific genes of choice consistently and efficiently has always been a major goal for scientists. The emerging field of RNA interference (*RNAi*), a process in which target mRNAs are degraded by small interfering RNA (*siRNA*), may indeed provide this long sought

after tool. The importance of this technology has been highlighted recently by Science, which has voted the *RNAi* discoveries as the "Breakthrough of the Year" in 2002. Essentially, *RNAi* involves an initiation and an effector step whereby introduced dsRNA is digested into 19-21 duplex *siRNA* by cleavage with Dicer and *siRNA* binds to an RNA-induced silencing complex (RISC). Activation of RISC targets the homologous sequence (transcript) and results in the cleavage of mRNA. The models of this *RNAi* mechanism and its applications, derived from biochemical and genetic approaches, are described in this minireview. So far, *RNAi* has proven to be a useful technique for genomic studies in C. elegans, D. melanogaster and various plants, for example. The use of *RNAi* has also been invaluable in studies in which morphological and developmental variability between species was investigated. Targeted, sequence specific siRNAs that suppress or silence gene expression have the potential to be in great demand as tools for for the *treatment* of human disease. There have already been several studies that have utilized *siRNA* to inhibit HIV-1 infection and replication, for example. The expansion of *RNAi* for biomedical therapeutics seems inevitable. This minireview analytically summarizes the advantages and the current and future potential of *RNAi* technology whilst simultaneously investigating any shortfalls or difficulties. Importantly, in vitro and in vivo applications in the laboratory and in human disease models are also...
MEDICAL DESCRIPTORS:
...virus replication; Human immunodeficiency virus 1; Human immunodeficiency virus infection--etiology--et; biomedicine; in vitro study ; in vivo study; disease model; gene function; protein degradation; *hepatitis C*--etiology--et; Hepatitis C virus; malignant neoplastic disease; cancer inhibition; human; nonhuman; human cell; animal cell; review; nucleotide sequence

5/3,K/17 (Item 1 from file: 94)
DIALOG(R)File 94:JICST-EPlus

06176693 JICST ACCESSION NUMBER: 05A0674190 FILE SEGMENT: JICST-E
Emerging drugs for chronic hepatitis C
BHOPALE GIRISH MAHADEORAO (1); NANDA RABINDRA KUMAR (1)
(1) Res. And Dev. Div., Hindustan Antibiotics Ltd., Pimpri, Pune 411018, Ind
Hepatol Res, 2005, VOL.32,NO.3, PAGE.146-153, FIG.1, TBL.1, REF.64
JOURNAL NUMBER: W1284AAX ISSN NO: 1386-6346
UNIVERSAL DECIMAL CLASSIFICATION: 615.281.8.03 615.281.8 616.9-08 578.1
LANGUAGE: English COUNTRY OF PUBLICATION: Japan
DOCUMENT TYPE: Journal
ARTICLE TYPE: Review article
MEDIA TYPE: Meta and P

...ABSTRACT: categories have already reached the different clinical phases of development. The present article highlights the status of current available therapies and emerging drugs for the *treatment* of hepatitis C. Copyright 2005 Elsevier B.V., Amsterdam. All rights reserved.
IDENTIFIERS: *siRNA*
BROADER DESCRIPTORS: *hepatitis C*...

5/3,K/18 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs

05209401 H.W. WILSON RECORD NUMBER: BGSA03209401
Interference of hepatitis C virus RNA replication by short interfering RNAs.
Kapadia, Sharookh B
Brideau-Andersen, Amy; Chisari, Francis V
Proceedings of the National Academy of Sciences of the United States of America v. 100 no4 (Feb. 18 2003) p. 2014-18
SPECIAL FEATURES: bibl f il ISSN: 0027-8424
LANGUAGE: English
COUNTRY OF PUBLICATION: United States

ABSTRACT: The effect of RNA interference (*RNAi*) on hepatitis C virus (HCV) replication was investigated. Current therapy of patients with chronic HCV infection includes *treatment* with IFNa in combination with ribavirin, but most *treated* patients do not resolve the infection, and, thus, an alternative therapy is crucial. Using a selectable subgenomic HCV replicon cell culture system, *RNAi* was found to specifically inhibit HCV RNA replication and protein expression in Huh-7 cells that stably replicate the HCV genome. This antiviral effect was independent of interferon, indicating that *RNAi* may represent a new strategy for the therapy of persistent HCV infection.
DESCRIPTORS:
Hepatitis C...

5/3,K/19 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

19620301 PMID: 16317665
***Treating* hepatitis C: can you teach old dogs new tricks?**
Rice Charles M; You Shihyun
Center for the Study of Hepatitis C Laboratory of Virology and Infectious Disease, The Rockefeller University, New York, NY, USA.
Hepatology (Baltimore, Md.) (United States) Dec 2005, 42 (6) p1455-8, ISSN 0270-9139--Print Journal Code: 8302946
Contract/Grant No.: AI40034; AI; NIAID; CA57973; CA; NCI
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

***Treating* hepatitis C: can you teach old dogs new tricks?**
... of the hepatitis C virus genome. CyPB interacted with the HCV RNA polymerase NS5B to directly stimulate its RNA binding activity. Both the RNA interference (*RNAi*)-mediated reduction of endogenous CyPB expression and the induced loss of NS5B binding to CyPB decreased the levels of HCV replication. Thus, CyPB functions as...
Descriptors: *Cyclophilins--physiology--PH; **Hepatitis C*--drug therapy --DT; *Peptidylprolyl Isomerase--physiology--PH; *Viral Nonstructural Proteins--physiology--PH

5/3,K/20 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

19420344 PMID: 16190563
[Experimental therapy in HCV infection]
Terapia eksperymentalna zakazenia HCV.

Inglot Malgorzata; Gladysz Andrzej; Rymer Weronika
Katedra i Klinika Chorob Zakaznych, Chorob Watroby i Nabytych Niedoborow Odpornosciowych AM we Wroclawiu.
Przegla d epidemiologiczny (Poland) 2005, 59 (2) p525-33, ISSN 0033-2100--Print Journal Code: 0413725
Publishing Model Print
Document type: Journal Article; Review ; English Abstract
Languages: POLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

PEGInterferon and ribavirin combination therapy is a gold standard in hepatitis C *treatment* . However it is not efficacious in all cases. Therefore, many studies are conducted to identifying additional drugs and therapeutic regimens, which might be more affordable...

... systems (e.g.replicon) is crucial for successful therapeutic intervention in viral life-cycle (viral NS5B polymerase and NS3/4A protease inhibitors, antisense nucleotides, ribozymes, *siRNA*).Other classes of immunomodulatory/antiviral agents and new interferon formulation have also been considered for IFN-based therapy also. On the other hand immunomodulatory pathways are attractive target for novel anti-HCV therapy. Combination therapy targeting different aspects will be probably in the future successful option in hepatitis C *treatment.*
Descriptors: *Antiviral Agents--therapeutic use--TU; *Hepacivirus; * *Hepatitis C*--drug therapy--DT; *RNA Replicase--antagonists and inhibitors --AI; *RNA, Viral--antagonists and inhibitors--AI; *Viral Nonstructural Proteins--antagonists and inhibitors--AI...; Humans; Interferon Alfa-2b --therapeutic use--TU; Pyrones--metabolism--ME; RNA, Catalytic--therapeutic use--TU; RNA, Small Interfering--therapeutic use--TU; Ribavirin --therapeutic use--TU; *Treatment* Outcome

5/3,K/21 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14201590 PMID: 12594341
RNA interference blocks gene expression and RNA synthesis from hepatitis C replicons propagated in human liver cells.
Wilson Joyce A; Jayasena Sumedha; Khvorova Anastasia; Sabatinos Sarah; Rodrigue-Gervais Ian Gael; Arya Sudha; Sarangi Farida; Harris-Brandts Marees; Beaulieu Sylvie; Richardson Christopher D
Ontario Cancer Institute, 620 University Avenue, Suite 706, Toronto, ON, Canada M5G 2C1.
Proceedings of the National Academy of Sciences of the United States of America (United States) Mar 4 2003, 100 (5) p2783-8, ISSN 0027-8424--Print Journal Code: 7505876
Publishing Model Print-Electronic
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

RNA interference represents an exciting new technology that could have therapeutic applications for the *treatment* of viral infections. Hepatitis C virus (HCV) is a major cause of chronic liver disease and affects >270 million individuals worldwide. The HCV genome is...
...functions as both a messenger RNA and replication template, making it an attractive target for the study of RNA interference. Double-stranded small interfering RNA (*siRNA*) molecules designed to target the HCV genome were

introduced through electroporation into a human hepatoma cell line (Huh-7) that contained an HCV subgenomic replicon. Two siRNAs dramatically reduced virus-specific protein expression and RNA synthesis to levels that were 90% less than those seen in cells *treated* with negative control siRNAs. These same siRNAs protected naive Huh-7 cells from challenge with HCV replicon RNA. *Treatment* of cells with synthetic *siRNA* was effective >72 h, but the duration of RNA interference could be extended beyond 3 weeks through stable expression of complementary strands of the interfering RNA by using a bicistronic expression vector. These results suggest that a gene-therapeutic approach with *siRNA* could ultimately be used to *treat* HCV.

Descriptors: **Hepatitis C*--metabolism--ME; *Liver--cytology--CY; *RNA --metabolism--ME; *RNA Interference; *RNA, Small Interfering--physiology --PH; *RNA, Viral--genetics--GE; *Virus Replication--physiology--PH

5/3,K/22 (Item 1 from file: 162)

DIALOG(R)File 162:Global Health

0005075949 CAB Accession Number: 20053130459

Experimental therapy in HCV infection.

Original Title: Terapia eksperymentalna zakazenia HCV.

Inglot, M.; Gadysz, A.; Rymer, W.

Katedra i Klinika Chorob Zakazonych, Chorob Watroby i Nabytych Niedoborow Odpornosciowych, Akademii Medycznej we Wrocawiu, ul. Koszarowa 5, 51-149 Wrocaw, Poland.

Conference Title: Jubileuszowe x Warsztaty Hepatologiczne. Polskiego Towarzystwa Epidemiologow I Lekarzy Chorob zakazonych oraz Polskiego Towarystwa Hepatologicznego. Jurata, Poland, 27-30 April 2005.

Przeglad Epidemiologiczny vol. 59 (2): p.525-533

Publication Year: 2005

ISSN: 0033-2100

Editors: Juszczyk, J.

Publisher: Panstwowy Zakad Higieny Warszawa, Poland

Language: Polish Summary Language: English Record Type: Abstract

Document Type: Journal article; Conference paper

Pegylated interferon (IFN) and ribavirin combination therapy is a gold standard in hepatitis C *treatment*. However it is not effective in all cases. Many studies have been conducted to identify new drugs and therapeutic regimens that are more affordable. The...

... g., replicon) is crucial for the successful therapeutic intervention in viral life cycle (viral NS5B polymerase and NS3/4A protease inhibitors, antisense nucleotides, ribozymes, and *siRNA*). Other classes of immunomodulatory/antiviral agents and new IFN formulation have also been considered for IFN-based therapy. On the other hand, immunomodulatory pathways are attractive target for novel anti-HCV therapy. Combination therapy targeting different aspects of the virus might be the future successful option for hepatitis C *treatment.*

...DESCRIPTORS: *hepatitis C*

5/3,K/23 (Item 2 from file: 162)

DIALOG(R)File 162:Global Health

0005037341 CAB Accession Number: 20053023541

The potential of RNA interference as a tool in the management of viral hepatitis.

Taylor, J. A.; Naoumov, N. V.

Author email address: n.naoumov@ucl.ac.uk

School of Biological Sciences, University of Auckland, Auckland, New Zealand.

Journal of Hepatology vol. 42 (1): p.139-144

Publication Year: 2005

ISSN: 0168-8278

Digital Object Identifier: 10.1016/j.jhep.2004.10.022

Publisher: Elsevier Amsterdam, Netherlands

Language: English Record Type: Abstract

Document Type: Journal article

The feasibility of using RNA interference (*RNAi*) to effect a potent and sustained replication is demonstrated and proof of concept of use of

RNAi as an antiviral therapy, particularly in the *treatment* of viral hepatitis (including hepatitis B, C and D) is offered. It is demonstrated that small interfering RNA has a higher degree of specificity in the *treatment* of human viral infections in comparison to conventional antiviral *treatment.*

...DESCRIPTORS: *hepatitis C*

5/3,K/24 (Item 3 from file: 162)

DIALOG(R)File 162:Global Health

0004993834 CAB Accession Number: 20043114474

RNA interference as a new strategy against viral hepatitis.

Radhakrishnan, S. K.; Layden, T. J.; Gartel, A. L.

Author email address: agartel@uic.edu

Department of Medicine, University of Illinois at Chicago, 840 S Wood Street, Chicago, IL 60612, USA.

Virology vol. 323 (2): p.173-181

Publication Year: 2004

ISSN: 0042-6822

Digital Object Identifier: 10.1016/j.virol.2004.02.021

Publisher: Elsevier Science San Diego, USA

Language: English Record Type: Abstract

Document Type: Journal article

Hepatitis viruses are the leading cause of liver cirrhosis and hepatocellular carcinoma worldwide. Since currently available *treatment* options against these viruses are limited, there is a need for development of alternative therapies. In this minireview, we concentrate on three hepatitis viruses - hepatitis C virus, hepatitis B virus, and hepatitis delta virus and discuss how RNA interference (*RNAi*) has been utilized against them. *RNAi* is a process by which small double-stranded RNA can effectively target a homologous RNA sequence for degradation by cellular ribonucleases. Though *RNAi* was exploited in the beginning for down-regulating cellular genes, it has recently been demonstrated that this process is equally effective against many types of human and animal viruses including the hepatitis viruses. Both synthetic small-interfering RNAs (siRNAs) and plasmid-based *siRNA* expression systems have been useful in suppressing the hepatitis viruses. Though this new approach looks promising, problems of nonspecific effects and delivery may need to be addressed before the full therapeutic potential of *RNAi* against viral infections in patients is realized.

...DESCRIPTORS: *hepatitis C*

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           83024  HEPATITIS C
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